Grand Rounds Review

Atopic Dermatitis in Children

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A 7-year-old girl presented with atopic dermatitis (AD) that did not respond to standard therapy. She was avoiding dairy, egg, and wheat in her diet because of a history of skin flares. Her weight gain was poor, and laboratory test results showed low iron and zinc levels. Over the previous 6 months, she had been prescribed numerous courses of antibiotics, but, despite this, she continued to have secondary skin infections as well as deep circumscribed erosions on her shins. She was awake much of the night because of scratching and displayed repetitive and habitual behavior. She also had troublesome allergic rhinoconjunctivitis with positive allergy testing results to house dust mite. Methicillin-resistant *Staphylococcus aureus* was isolated from her skin, which was successfully treated with appropriate antibiotics and flares controlled with topical antiseptics and better personal and caregiver hygiene. Although milk, egg, and wheat specific IgE were raised, these foods were successfully reintroduced back into her diet with improvement of her nutritional status and no flare of her AD. In view of her habitual behavior and family history of obsessive compulsive disorder, she underwent cognitive behavioral therapy, and her general well-being, sleep, and ulcers over her shins improved. Despite high house dust mite–specific IgE, house dust mite sublingual immunotherapy led to no additional improvement in her AD although it did improve her rhinitis. Although there may be no "quick fixes" in patients with AD, the clinician should be aware of antimicrobial, allergen, and educational and/or behavioral interventions, which may greatly improve eczema severity and the patient's well-being. **(J Allergy Clin Immunol Pract 2014;■:■-■)**

Key words: *Atopic dermatitis; Food allergy;* Staphylococcus aureus*; MRSA; Zinc; Hyper IgE syndrome*

A Pakistani girl initially presented to the specialist pediatric allergy and atopic dermatitis (AD) outpatient clinic at 7 years old with worsening AD not controlled with moisturizers and moderately potent topical corticosteroids (clobetasone butyrate). Her AD had started at 2 months old, but, until 6 months ago, it had been confined largely to the flexural surfaces of her elbows and knees. According to her mother, during early childhood her AD had flared with cow's milk, egg, and wheat, and she was avoiding these foods as well as nuts. In place of cow's milk she was consuming soybean-based substitutes with vitamin D supplements. Six months before being referred, her skin became impetiginized and painful. She had been treated almost continuously with β-lactam antibiotics that had staphylococcal coverage because her skin flared within a week of finishing an antibiotic course. At the initial consultation, there were areas with crusting and oozing.

Methicillin-resistant *Staphylococcus aureus* (MRSA) was isolated from skin swabs. Treatment with 2-week courses of oral clindamycin settled the secondary infection. She slept in a 1-bedroom house with her 2 younger sisters and parents, none of whom had AD or skin infections. The family kept no pets. The patient and her family were educated regarding cross- and re-infection. Antiseptic hand washes and bath additives that contained chlorhexidine were prescribed to reduce the bacterial load on the skin. Infective flares were subsequently much less frequent and occurred only once or twice a year. On one occasion, when a vesicular rash was noted, herpes simplex I was isolated from vesicular fluid by PCR, and she received a 5-day course of acyclovir. Her AD remained controlled with regular use of dry cotton bandages and by changing from the moderate topical corticosteroid to a calcineurin inhibitor (tacrolimus 0.03% ointment twice a day).

As well as AD, dairy products and possibly wheat were said to have caused bloating and loose bloody stools when she was an infant. On testing, stool results were negative for reducing substances. Antitransglutaminase autoantibody results were also negative, and upper and lower endoscopy results were normal. Her total serum IgE was 5800 kU/L (reference value, <100 kU/L). Skin prick tests and specific IgE were positive for cow's milk (32 kIU/L), egg (12 kIU/L), wheat (43 kIU/L), peanut (>100 kIU/L), and house dust mite (*Dermatophagoides pteronyssinus* and *Dermatophagoides farnae*) (both >100 kIU/L). The patient also was assessed by a pediatric dietitian because she appeared thin, with her weight on the fourth percentile and height on the 25th percentile for her age. Results of blood tests showed no anemia, but a microcytosis, low serum ferritin (17 μg/L [30-350]) and zinc concentrations (9.0 μmol/L [10-18]). Plasma calcium, phosphate, and vitamin D levels were normal. Cow's milk, egg, and wheat were all successfully reintroduced into her diet after formal oral challenges in the hospital with no acute allergic reactions, flaring of her eczema, or gastrointestinal symptoms. Repeated endoscopy performed after 6 months on a cow's milk– and wheat-containing diet was normal. With dietary advice and calorie, iron, and zinc supplements, her weight improved, and she grew along the 25th percentile for both her weight and height. Formal food challenge

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Abbreviations used
AD- Atopic dermatitis
MRSA- Methicillin-resistant Staphylococcus aureus

to peanut under medical supervision resulted in an urticarial rash and bronchospasm after ingestion of 1 g of peanut. The patient was provided with an allergy management plan and an epinephrine autoinjector.

Despite the improvement in her AD, which, with the above treatment, was largely confined to her wrists and ankles, she continued to be troubled with pruritus, particularly at night and averaged only 2 to 4 hours of sleep, even with the use of a sedative antihistamine. At the age of 11 years, she developed deep circumscribed areas of excoriation and ulceration on the anterior aspect of her shins. It was apparent that she was increasingly obsessed with hand washing, which often went on for 30 minutes after visiting the bathroom. Her sleep disturbances were made worse by her habitual cleaning behavior, removing dead skin from the bed. Her father and paternal grandfather also had excessive habitual washing. The patient was referred to the regional child and adolescent psychiatry service for cognitive behavioral therapy. With this therapy, her general well-being, sleep pattern, and, particularly, the excoriations over her shins improved, although her skin remained very friable.

As well as her AD, she also developed allergic asthma and perennial rhinitis poorly controlled with inhaled steroids (fluticasone propionate 100 mg twice a day) and regular oral antihistamines and nasal steroids (fluticasone furoate 55 mg once a day). Montelukast was tried but was not tolerated because of worsening insomnia. Because of high *Dermatophagoides pteronyssinus*– and *Dermatophagoides farnae*–specific IgE, house dust mite sublingual immunotherapy was commenced at the age of 10 years. It made little difference to her AD, but her asthma or allergic rhinitis improved in terms of symptoms, quality of life scores, or medication use. Her allergic diseases remain relatively well controlled, flaring sometimes when she misses doses of her medication.

FACTORS TO CONSIDER WHEN AD FLARES

Secondary bacterial infection

Many parents of children with AD come to their first consultation with the physician anxious to have allergy tests to find a possible allergic trigger. However, particularly when there are signs of secondary skin infection (crusting, oozing, and pain), the trigger is more likely to be secondary bacterial skin infection, typically with *Staphylococcus aureus*. In older children and adults whose diet has not changed but their AD has suddenly flared, infection rather than food allergy is the leading cause of exacerbation.

It is important to inquire about previous antibiotic use, as in patients who have had multiple courses of antibiotics and response is only transient, MRSA should be considered and swabs taken to guide appropriate antibiotic use. In patients with AD, MRSA can either be acquired within the community or otherwise from caregivers working in health care settings or from relatives who had recently been in the hospital. Most MRSA strains express additional virulence factors and/or exotoxins.[1] Living conditions should also be considered because the patient's home environment may promote cross- and re-infection. This is more likely where living conditions are cramped, as in this case, or where the family has pets, which may transfer bacterial pathogens. Although less common, a vesicular rash suggests superadded infection with the herpes simplex virus–eczema herpeticum, which can be associated with disseminated viremia and, as such, warrants prompt treatment with acyclovir and inpatient care.

Food allergy

Exacerbation of AD by foods is classically a delayed T-cell–rather than immediate IgE-mediated hypersensitivity reaction. The latter would present as urticaria or angioedema. Common foods that cause flares in AD are cow's milk protein, egg, soybean, and wheat.[2] Immediate hypersensitivity reactions are more common in children with AD and may be present in conjunction with delayed reactions, as in this case, in which the patient also had a peanut allergy. Total IgE concentrations are frequently high in patients with AD, which leads to clinically nonrelevant positive allergen-specific IgE.[3] Allergy tests should not be used as the sole determinants of dietary restrictions. In addition, high total IgE should not be confused with hyper IgE syndrome, in which the key clinical features are (1) severe or recurrent pyogenic infections, particularly of the chest, associated with bronchiectasis and/or pneumatoceles; (2) a family history because the most common form is inherited in an autosomal dominant pattern; (3) fractures with minimal trauma; and (4) delayed shedding of primary dentition.[4-6] Causes of raised total serum IgE are summarized in Table I.

Assessment of the child's growth is important because particularly those with dietary restrictions may have signs of failure to thrive or even rickets, which need additional supplements and input from a dietitian. Gut-associated immunologic disorders such as celiac disease (Th1 mediated) and eosinophilic enteropathies (Th2 mediated) should also be considered in patients who are failing to thrive or have prominent gastrointestinal symptoms. Both eosinophilic esophagitis and celiac disease are more common in patients with AD.[7,8] In a large cohort of 620 patients with eosinophilic esophagitis, 13% had concomitant AD,[9] and, in 173 adults with celiac disease, AD was found to be 3 times more common than in close relatives who did not have AD. Clinical features of eosinophilic enteropathies depend on the age of the child and the segment of the gut involved. They include failure to thrive, dysphagia, vomiting, abdominal pain, and/or diarrhea.

Inherited disorders of zinc metabolism or nutritional zinc deficiency that leads to acrodermatitis enteropathica should be considered in children with prominent perioral and perianal erythema.[10] Younger children also may have diarrhea and behavioral changes. Older children display failure to thrive, alopecia, nail dystrophy, and recurrent infections. In these patients, plasma zinc concentrations are low and the dermatitis responds after 5 to 10 days of zinc supplements. In patients with an inherited disorder, lifelong zinc supplements are required. Although serum ferritin may be low in children with AD, there is no evidence that supplementary iron improves AD symptoms.

Social and psychological considerations

Pruritus is an essential feature of children with AD. It can be particularly troublesome at night time when the patient is less distracted. The rash of AD is exacerbated by scratching, and, in some patients, as in the case presented, excoriation and severe

TABLE I. Causes of high total serum IgE concentrations

Cause	Comment
AD	Most common cause of high total IgE concentrations in children, typically >1000 kU/L but may occasionally be >100,000 kU/L
Other allergic conditions	Allergic asthma and rhinitis, drug allergies, bronchopulmonary aspergillosis
Parasitic infections	Cestodes (eg, echinococcosis) trematodes (eg, schistosomiasis), nematodes (eg, ascariasis, toxocariasis)
Hyper IgE syndrome (rare)	Autosomal dominant (*STAT3* gene mutation) and recessive (*TYK2* and *DOCK8* gene mutations) forms; although dermatitis may occur, key features are lung infections with bronchiectasis and pneumatoceles, delayed shedding of teeth and fractures; key features of the autosomal recessive form are viral infections, including eczema herpeticum, molluscum contagiosum, varicella zoster virus and human papilloma virus infections, bacterial sinopulmonary infections and impetigo, dermatitis, and food allergies
Other T-cell immunodeficiency diseases (rare)	Wiskott-Aldrich syndrome (*WASP* gene mutation) associated with thrombocytopenia and hemorrhage, sinopulmonary infections, autoimmunity associated with EBV infection; Omenn syndrome (numerous genetic causes) a leaky form of severe combined immunodeficiency associated with erythroderma and death from overwhelming viral, bacterial, and fungal infections
Autoimmune or neoplastic conditions	Polyarteritis nodosa; IgE monoclonal gammopathy

damage can occur. When there is extensive excoriation, erosions, or demarcated ulcers, alternative or concomitant skin conditions such as dermatitis artefacta should be considered. Dermatitis artefacta is defined as self-inflicted skin damage to satisfy an unconscious psychological or emotional need. There may be many reasons for this in children (Table II). In patients with refractory AD, it is important to consider social and psychological factors that may act as potential stressors in the home, school, or work environment.

TABLE II. Trigger factors that lead to excessive skin damage in children with AD

Cause	Example
Perceived or real neglect	Birth of brother or sister; sibling with a condition that needs excessive attention; parental separation that leads to changes in focus of mother and father; significant life event in family member: death, accident, serious illness, etc
Physical or emotional abuse	Physical abuse, bullying at school, racial abuse, sexual abuse
Psychological disorders	Emotional disorder—anxiety (personal stress or stress within the family or friends, eg, death of grandparent, serious illness of best friend) or depression; obsessive compulsive disorder

Nonadherence

Nonadherence is common in patients with chronic diseases. In children with AD, nonadherence with topical therapy may occur in as many as two-thirds of patients.[11] There are various reasons for this (Table III).[12] Poor adherence and concerns about adverse effects are a cause of treatment failures and should be considered in all patients with refractory AD.[13,14] Nonadherence because of false concerns about conventional therapy can seriously affect the quality of life of patients. It is the responsibility of the physician to ensure that nonadherence does not lead to neglect or, in rare cases, death.[15]

Natural clinical variability with waxing and waning

AD is a disease that may fluctuate in severity for no obvious reason, with both eczema flares and associated urticaria. The inability to explain these flares often prompts the patient to seek advice, sometimes from multiple health care workers, which leads to conflicting treatment plans. In many patients with moderate-to-severe AD, the answer may be apparent at the initial greeting. A genetically inherited mutation in the filaggrin (*FLG*) gene that leads to leaky skin barrier function is common, particularly in patients of Northern European and Far East ethnicity.[16] Over a 2-year period, we screened 113 children who attended our specialist pediatric allergy/AD practice and found that 40% to 50% of children with moderate-to-severe AD had *FLG* mutations (R501X or 2282del4); 19% were homozygous or compound heterozygous. Hand eczema was a strong clinical marker of mutations, but even more discriminatory was palmar hyperlinearity (Figures 1 and 2).[17] By using area under receiver operator curve (AUROC) analysis, palmar hyperlinearity was highly discriminatory particularly for patients with homozygous *FLG* mutations (AUROC 0.86 (0.75-0.97); $P < .001$). *FLG* mutations are also associated with an increased risk of eczema herpeticum.[18]

TREATMENT OPTIONS

Management of skin barrier dysfunction

Educating the patient and parents regarding the natural history of AD; empowering them how to keep the disease under control, prevent, and control flares; and the risk-benefit of the various treatment options are key to adherence and symptom control.[19,20] Particularly when the disease is troublesome and impacts on the quality of life of the patient and his or her family,

TABLE III. Causes of nonadherence with treatment in patients with AD

Cause	Examples	Methods of improving adherence
Patient's or parent's misperception of disease	Lack of understanding about the disease; conflicting information received from multiple independent health care and allied professionals	Education regarding disease process; use the literature and ongoing community support; practical explanations, demonstrating correct use, and application of treatments; treatment action plan; early intervention and support
Child resisting treatment	Confrontation between parent and child	
Patient's or caregiver's perceived or real concerns regarding medication	Patient perceives that medication is not effective; medication stings or hurts when applied; perceived risk of adverse effects, eg, steroid phobia; lack of understanding of the need for ongoing therapy; patient or caregiver has insufficient time in daily routine to apply medication	Allow patients and caregivers to express and discuss their concerns; individualize management plans to accommodate specific needs, which allows for daily variability; educate and train patients and/or caregivers; information sheets; educate and train health care professionals; ongoing support; reduce frequency of application and/or number of ointments used; involve extended family members
Medication unavailable	Patient or caregiver fails to go to physician or pharmacist for prescription renewal; physician prescribes too little medication; physician refused to prescribe medication because of cost or perceived risks	Provide caregivers and prescribers with information regarding amount of ointment required each week

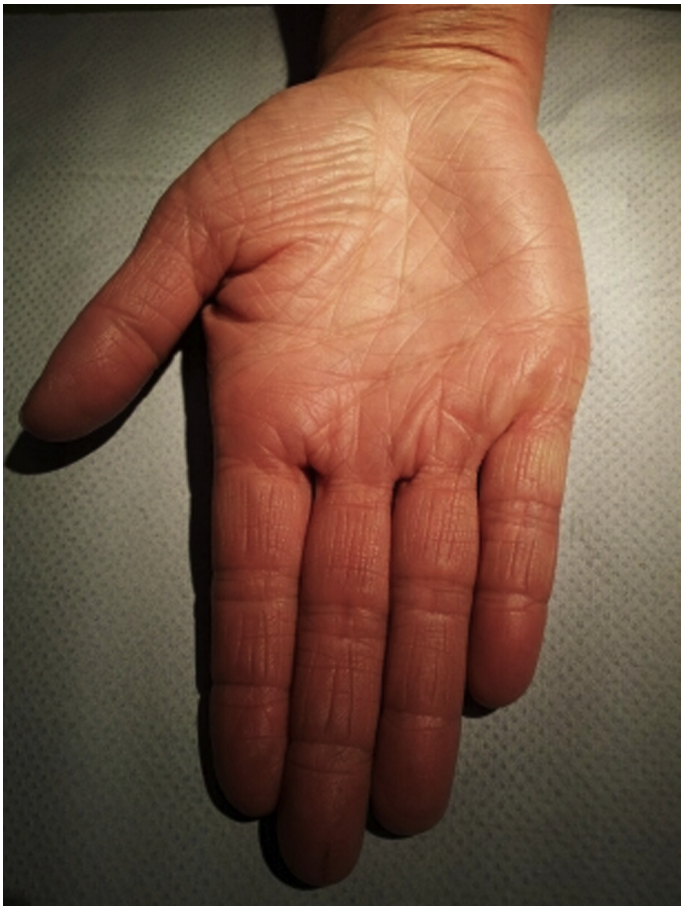

FIGURE 1. A patient with evidence of palmar hyperlinearity and an underlying *FLG* gene mutation.

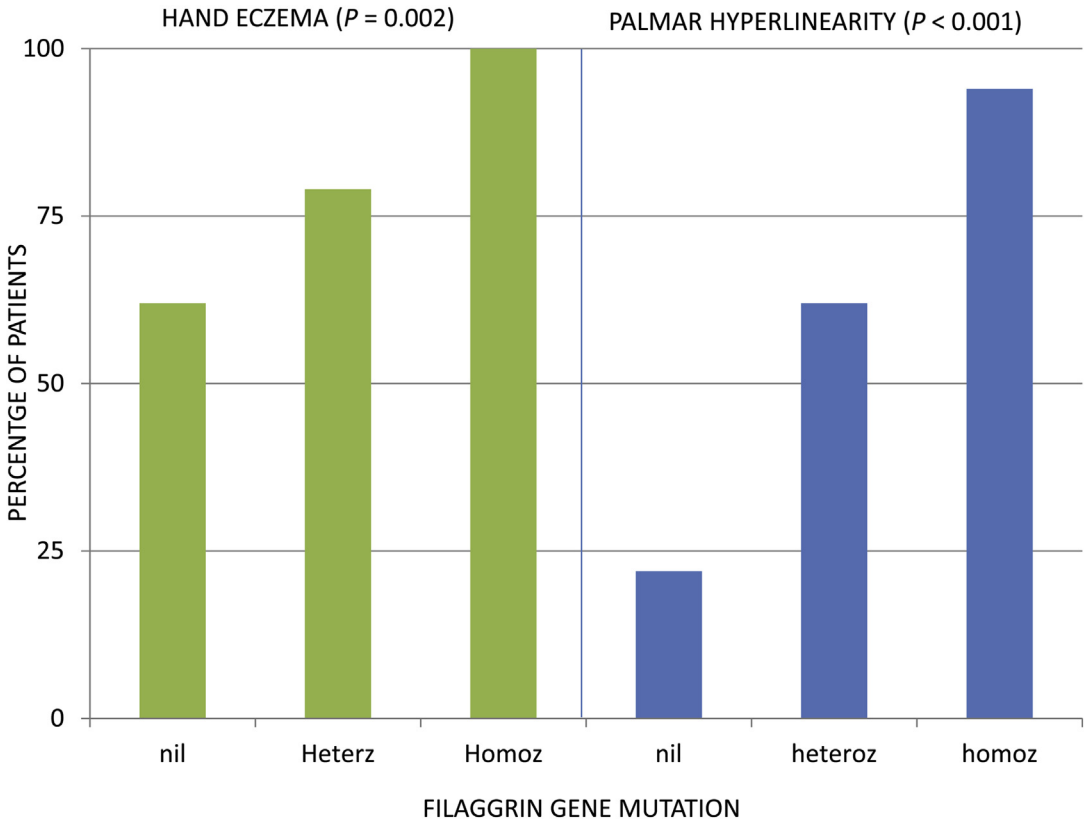

FIGURE 2. Association of confirmed *FLG* gene mutation, hand eczema, and palmar hyperlinearity in a cohort of 113 children with moderate-to-severe disease who attended a specialist allergy/AD clinic. *Heteroz,* Heterozygous *FLG* mutations; *Homoz,* homozygous or compound heterozygous *FLG* mutation.

consultation with the physician can be supplemented by (1) written information and management plans, (2) support from allied health care workers in the clinic or community, and (3) advice from patient support groups.[20,21] Realization that an inherited (eg, *FLG* gene mutations) or acquired defect in skin barrier function is an important contributory factor in a proportion of patients with AD should focus treatment on optimizing skin moisturization and avoiding soaps and fabrics that may further disrupt the skin barrier. Appropriate use of long-sleeve clothes, cotton and/or silk undergarments to help prevent exposure to environmental allergens and irritants, and damage from scratching also can be helpful. Although antihistamines do not relieve the itch of AD, they may control concomitant urticaria in some patients. Sedative antihistamines also may promote sleep when used at bedtime.

Topical immunosuppression

Topical corticosteroids remain seminal in the treatment of AD. In children, a mild potency steroid, for example, 1% hydrocortisone ointment used up to twice a day, is recommended when the disease is not controlled with moisturizers alone. Contrary to common belief, there is little objective evidence that long-term use of topical corticosteroids causes chronic skin atrophy, adrenal suppression, growth retardation, or reduced bone mineral density.[22,23] However, it generally is recommended that, particularly in children, the lowest potency topical steroid that effectively controls the disease is used. In children older than 2 years old, particularly when there is facial involvement and inadequate control with a mild potency steroid, a calcineurin antagonist might be considered after consultation with a clinician experienced in the use of these drugs.[24] In 2005, because of the absence of safety data to show no risk of cancer from topical calcineurin inhibitors for AD, a black box warning was issued by the U.S. Food and Drug Administration. A long-term international safety study is currently in progress, and there remains no evidence to suggest a significant association.[25,26] In contrast, AD *per se* as well as long-term use of potent topical corticosteroids have been associated with a 2-fold risk of lymphoma. The exact nature of this association remains to be determined.[24-26]

Management of secondary skin infections

For patients with whom there are clinical signs of secondary skin infection, particularly in those who have had repeated courses of antibiotics, skin swabs should be taken to confirm the bacterial pathogen and antibiotic sensitivities. Although there are increasing worldwide concerns regarding community-acquired MRSA,[27] these strains seem to be relatively uncommon in patients with AD ($\leq$2% in Toronto, Canada,[28] San Diego, California,[29] Porto Alegre, Brazil,[30] and Hamilton, New Zealand[31]). In contrast, older children who attend specialist centers and who are exposed to repeated courses of antibiotics may have much higher rates of MRSA, which probably relate more directly to prolonged exposure to anti-staphylococcal antibiotics (up to 19% in Manchester,

TABLE IV. Summary of key clinical features and treatment options of patients with severe or refractory AD poorly responsive to regular moisturizer and mild potency topical corticosteroid

Clinical features	Diagnosis/investigations	Treatment
AD localized to exposed areas; hand eczema; palmar hyperlinearity; family history, parent with same clinical features	Poor skin barrier function; *FLG* gene mutation screen if available (currently largely confined to the research setting)	Optimize skin barrier: moisturizers, cotton or silk garments, long-sleeve clothes; if concomitant intermittent urticaria, then consider long-acting, nonsedating antihistamine; avoid irritants and/or allergens if possible
Crusting, oozing, painful lesions	Secondary skin infection: bacterial and/or viral; check antibiotic history; skin swabs; if vesicular send fluid for viral PCR	Appropriate antibiotics based on culture sensitivities and local policies; if vesicular, then consider hospital admission for intravenous acyclovir; topical antimicrobial soap substitute and bath additives; if generalized, severe and once infection is under control, consider oral cyclosporine under specialist supervision
Excessive excoriation and/or ulceration	Underlying psychological and/or social stress; ask about home, school, work environment, significant life events; history of behavioral and/or psychiatric disturbances	Address any stressor if identified, may need help of community allied health professional, school nurse, social worker, psychologist, or psychiatrist; behavior therapy
History of exacerbation by foods, dietary restrictions, poor weight gain, gastrointestinal symptoms; moderate-to-severe generalized erythrodermic disease in infants and young children	Investigate for food allergy with 4 wk of food avoidance and then rechallenge; avoid dietary manipulation based solely on allergy tests; consider concomitant celiac disease or eosinophilic enteropathies	Avoid foods that objectively cause flares; reintroduce foods that are tolerated; ensure suitable calorie, vitamin, mineral supplements; engage dietitian, particularly if dairy or wheat; plan for an intermittent trial of reintroduction of foods at 6- to 12-mo intervals
History of flares with topical application of creams, ointments, washes or sprays; asymmetrical distribution	Secondary contact AD; consider patch testing*	Avoid specific chemicals

*Data from reference 51.

United Kingdom[32]). When the rash is vesicular, vesicular fluid rather than just a swab should be taken in a specific viral medium.

Antibiotics should be prescribed based on culture sensitivities and regional antibiotic policy.[32,33] Except for the most severe extensive cases, a course of oral antibiotics is usually effective. If the infection is extensive, particularly if on the face, when the patient is systemically unwell with fever, or when the antibiotic can only be given intravenously, then inpatient treatment is required. Although antibiotics are typically prescribed for 5 to 7 days, more prolonged courses of up to 2 weeks may be required to clear the clinical infection, although it is unlikely that the *S aureus* will be completely eradicated from the skin. Diagnosis of eczema herpeticum is often an indication for inpatient therapy with intravenous acyclovir.[34]

Topical antimicrobial hand, shower, and bath solutions should be considered for both the patient and other members of the family who live at home to reduce skin carriage and cross-infection. Eradication of nasal carriage by using topical antimicrobial antiseptic ointments might also be prescribed based on regional policy. "Spring cleaning" the patient's bedroom, washing and replacing the linen, and wiping down surfaces and toys may help to reduce re-infection from fomites. Tubs and tubes of moisturizers and ointments may need replacing if there is a risk of bacterial contamination.[35]

Management of food allergy

Foods that cause AD to flare are an important part of young children's diet. Early input from a dietitian should be sought to ensure that (1) the food allergen is completely avoided, (2) calorie intake is optimized, and (3) vitamin and mineral deficiencies, including rickets, do not develop. In the case presented, not only were cow's milk and wheat specific IgE predictive of sensitization rather than clinical allergy as is often the case in food-associated AD flares, but the patient was failing to thrive and had deficiencies in mineral intake because of unnecessary dietary restrictions. If food-induced AD flares are suspected, then a 4-week period of dietary avoidance should generally be followed by a trial of reintroduction of the food to confirm the diagnosis. Proven allergies to cow's milk, egg, wheat, and soybean often resolve with age, and so trying to reintroduce the food at usually 6- to 12-month intervals to determine if tolerance has developed is important. Celiac disease can often be ruled out with an antitransglutaminase test, but the test may be negative if wheat is being excluded, in which case the test will need to be repeated after wheat has been reintroduced. In some children, assessment by a gastroenterologist will be required to rule out eosinophilic enteropathies.

Management of psychological stressors

Assessment of the home and school environment is useful in identifying trigger factors. Support by nurses, social workers, and psychologists in the community may be required to manage the patient's condition. In the case presented, the child's shin ulceration, which was due to her obsessive-compulsive tendencies, improved after referral to the local psychiatry service and cognitive behavioral therapy.[36,37] In other cases, biofeedback and

relaxation therapy; medication; or, when there is evidence of risk of self-mutilation or suicide, hospitalization may be required. Identifying and dealing with a school bully, recent bereavement, or racial abuse may lead to amelioration or even resolution of some patients' AD. Community health care workers and allied health care professionals can provide support and education, and can reinforce management plans.[38,39] They also can address ongoing concerns and deal with intermittent flares, which may otherwise lead to disillusionment followed by nonadherence and treatment failure, and result in poorer quality of life.[40]

Systemic immunosuppression

Systemic immunosuppressants should only be used by physicians experienced in their use. The risks, particularly of serious infection, for example, chickenpox or eczema herpeticum, must be discussed with patients before starting therapy. Although oral corticosteroids are effective in the short term, their use should be discouraged because of the risk of flares after discontinuing therapy and serious long-term adverse effects. Oral cyclosporine can ameliorate generalized flares due to bacterial skin infection and secondary superantigen-induced polyclonal T-cell activation.[41,42] It is important to initially treat the infection with appropriate antimicrobials. Azathioprine also may be beneficial in some patients with severe disease, although the evidence for use of this and other immunosuppressants is largely anecdotal. Phototherapy also may provide some relief, but effects are usually transient.

Omalizumab is effective in ameliorating allergic asthma and rhinitis as well as chronic urticaria.[43] Although there are a number of case reports and open-labelled studies, there is little evidence from a placebo-controlled, double-blinded clinical trial to show that omalizumab is effective in AD.[44] One underpowered study of 48 children with AD suggested that house dust mite sublingual immunotherapy may lead to an improvement in a subset of patients.[45,46] Appropriately powered multicenter randomized, double-blinded, placebo-controlled trials are required before either of these therapies can be recommended in routine clinical practice. Time will tell whether biologics that upregulate filaggrin expression or block the initiation of the Th2-immune response by skin epithelium, for example, anti-thymic stromal lymphopoietin antibodies may herald a new era in the management of severe AD.[47,48]

SUMMARY

The medical management of all chronic diseases are confounded by the fact that patients and caregivers are frustrated by the poorer quality of life and continuing need for regular medication. It is human nature to search for the elusive "quick fix," either from health care professionals or alternative medical practitioners. Although the quick fix may not be possible in all patients with AD, individualizing care based on identifying and treating key triggers can lead to amelioration and sometimes even remission of this common multifactorial atopic disease (Table IV).[49] Physicians also play an important role in providing advice, support, and reassurance to patients and parents in the management of this chronic disease.[50]

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